29. The method of claim 24, wherein one or more of the amino acids residues has been exchanged with a residue of an amino acid having similar size, charge and polarity, or with amino acid mimetics resulting in one or more backbone modifications.

1

30. The method of claim 24, wherein said part does not contain one or more sections of 5-30 amino acids corresponding to T cell epitopes of said microbial protein, the T cell which epitope of said microbial protein having less than 4 consecutive amino acids which are identical with the corresponding amino acids of said mammalian stress protein amino acids, such that said peptide includes a microbial T cell epitope having sufficient sequence identity with a T cell epitope of said mammalian stress protein homologue and lacks any microbial T cell epitope which does not have sufficient sequence identity with corresponding amino acids of said mammalian stress protein homologue.

REMARKS

This is in response to Office Action dated July 25, 2000. Reconsideration is requested.

In order better to define the essence of the present invention, new claims 24-30 are submitted herewith directed to the method of treatment of or protection against an inflammatory disease by administering a peptide active agent according to the present invention. Support for the method claims is found throughout the specification, for example, at page 4, last full paragraph.

The aggregated prior art, including but not limited to the Munk et al. and Quayle et al. references of record, does not suggest anything like the use of the present peptide(s) for anti-inflammatory or autoimmune disease prevention or treatment. Indeed, in the second to the

last sentence of the abstract of Ouayle et al., the authors advise that "Our findings, therefore, do not disagree with the postulate that autoimmune disease could possibly be triggered by bacterial epitopes with homology to self protein." In their belief that peptides generally similar to those of the present application would trigger autoimmune diseases, Quayle et al. not only completely missed the present invention, but taught away from it. In contrast to Quayle et al., the present Applicants discovered that the peptides of the present invention have a protective and/or therapeutic effect on inflammatory diseases and autoimmune diseases. Munk et al. do not address the present medical indications. Because the prior art does not teach the claimed method and actually teaches away from it, the claims have been shown to be non-obvious, novel and patentable.

Entry of new claims 24-30 and allowance of the above-identified patent application are respectfully requested.

Respectfully submitted,

WEBB ZIESENHEIM LOGSDON ORKIN & HANSON, P.C.

Barbara E. Johnson

Registration No. 31,198

Attorney for Applicants 700 Koppers Building

436 Seventh Avenue

Pittsburgh, PA 15219-1818

Telephone: (412) 471-8815 Facsimile: (412) 471-4094